DRAFT -- JANUARY 30, 1992

IV.

UNITED STATES DEPARTMENT OF LABOR
OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION

COMMENTS OF
THE TOBACCO INSTITUTE

ON

PRIVILEGED & CONFIDENTIAL

OCCUPATIONAL EXPOSURE TO INDOOR AIR POLLUTANTS:
REQUEST FOR INFORMATION

Docket No. H-122, RIN 1218-AB37

DRAFT

March 20, 1992

2024695648

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

TABLE OF CONTENTS

		PAGE
I.	SCIENTIFIC RESEARCH ON EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE SHOWS NO SIGNIFICANT RISK TO HUMAN HEALTH	3
	A. The Composition of Environmental Tobacco Smoke	3
	B. ETS Levels in the Workplace	7
	C. The Alleged Health Effects of ETS	10
	1. Lung Cancer	10
	a. Epidemiologic Studies	10
	b. Meta-Analysis of ETS Exposure Studies	19
	2. Cardiovascular Disease	27
	3. Respiratory Effects on Healthy Adults	33
	4. Respiratory Effects on Sensitive Individuals	34
	5. ETS and Sick Building Syndrome	36
	6. Irritating Effects	39
II.	ETS EXPOSURE IN THE WORKPLACE IS NOT ASSOCIATED WITH A "SIGNIFICANT RISK" OF MATERIAL IMPAIRMENT OF HEALTH	40
	A. The Legal Standard	40
	B. The Available Evidence Does Not Support a Finding of Significant Risk	43
	1. Inadequate Evidence of Significant Risk	44
	2. Insufficiency of Workplace Exposure Data	45
	3. Inadequate Dose-Response Information	46
	C. OSHA Cannot Rely Upon Data Extrapolated From Other Sources To Support Regulation of ETS	49
	D. Mere Irritation or Annoyance Does Not Constitute a "Material Impairment of Health"	54
III.	CONCLUSION	55

2024695649

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

OCCUPATIONAL EXPOSURE TO INDOOR AIR POLLUTANTS:

REQUEST FOR INFORMATION

Docket No. H-122

January 21, 1992

These comments respond to the Request for Information on Occupational Exposure to Indoor Air Pollutants (the "RFI") and are submitted by The Tobacco Institute, which represents major United States manufacturers of tobacco products. These comments respond to the issues raised by the RFI concerning occupational exposure to "passive tobacco smoke," also known as environmental tobacco smoke ("ETS"). We also have asked a number of independent scientists and ventilation specialists to provide additional comments.

In order to promulgate a standard regulating workplace exposure to a particular substance, the Occupational Safety and Health Act requires OSHA to prove that the regulation is reasonably necessary and appropriate to remedy a significant risk of material health impairment. 29 U.S.C. § 652(8), 655(b)(5); Industrial Union Department, AFL-CIO v. American Petroleum Institute, 448 U.S. 607, 639 (1980). A finding of significant risk must be supported by substantial evidence in the record considered as a whole. 29 U.S.C. § 655(f); AFL-CIO v. Marshall, 617 F.2d 636, 649-50 (D.C. Cir, 1979).

As these comments show, there are inadequate data to suggest that exposure to ETS is associated with an increased

2024695650

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

risk of lung cancer or other disease in nonsmokers and, thus, insufficient justification for OSHA to regulate exposure to ETS on this basis. Moreover, there are few data focusing on ETS exposure in the workplace; the available epidemiologic data do not support a statistically significant association between occupational exposure to ETS and an increased risk of lung cancer or other disease. ETS is only one factor affecting indoor air quality and scientific data suggest that ETS is typically a minor contributor to indoor air quality problems. In fact, a standard that simply banned smoking could create a false sense of security because tobacco smoke is often the only visible sign of inadequate ventilation. If OSHA believes that regulation of indoor air quality is necessary, a standard requiring adequate and well-maintained ventilation systems would address both occupant complaints about ETS and a variety of indoor air problems.

In November 1989, an International Symposium on Environmental Tobacco Smoke was held at McGill University in Montreal, Canada.[1/] Over seventy scientists from throughout the world participated in the conference. Participants included experts in chemistry, medicine, toxicology, epidemiology, statistics, risk assessment and indoor air quality. Scientific presentations and panel discussions

---

[1/] Environmental Tobacco Smoke (1990). Proceedings of the International Symposium at McGill University, Ecobichon, D.J. and Wu, J.M. (eds.), Lexington Books, Lexington, MA, at 333-356. [hereinafter cited as McGill Symposium]

2024695651

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

comprehensively evaluated the available scientific data on ETS. The conference organizers concluded that the published data, when critically examined and evaluated, do not support the claim that ETS is a health hazard. Some participants noted in particular the lack of evidence of a significant risk from workplace ETS exposure and stressed that ETS is not synonymous with indoor air pollution and that regulation of smoking will not ensure improvements in indoor air quality. The published proceedings of the McGill Symposium are submitted with these comments for the rulemaking record. As will be shown below, no study or information has become available during the past 14 months that would alter the conclusions of the McGill Symposium.

I. SCIENTIFIC RESEARCH ON EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE SHOWS NO SIGNIFICANT RISK TO HUMAN HEALTH

A. The Composition of Environmental Tobacco Smoke

Environmental tobacco smoke ("ETS") is a highly dilute mixture of exhaled mainstream and sidestream smoke that decays quickly after initial formation, depending on ventilation and other factors.[2/] A considerable amount of scientific research has been conducted during the last decade

---

[2/] Gori, G. and Mantel, N. (1991). Mainstream and environmental tobacco smoke, Reg. Tox. and Pharm., 14: 88-105; Baker, R. and Proctor, C. (1990). The origins and properties of environmental tobacco smoke, Environ. Int., 16: 231-245.

2024695652

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

in an attempt to characterize the composition of ETS.[3/] Nevertheless, few of ETS's constituents have been identified and relatively little is known about the precise chemical and physical changes that occur as ETS forms, ages and decays. The complex nature of ETS, and the many unresolved questions that result, must be taken into account in considering whether exposure to ETS in the workplace is associated with a significant risk of disease in nonsmokers.

At present, this much is known about ETS:[4/]

1. ETS originates from a combination of exhaled mainstream smoke (the smoke that is exhaled after a smoker draws a puff) and sidestream smoke (the smoke that is emitted from the tip of a burning cigarette or is diffused through the cigarette paper).[5/] From formation to disappearance, ETS constantly changes in concentration, relative composition and in the phase distribution of its constituents.[6/]

[3/] Eatough, D., et al. (1990). The chemical characterization of environmental tobacco smoke, McGill Symposium at 3-39; Guerin, M. (1987). Formation and physiochemical nature of sidestream smoke, Environmental Carcinogen: Methods of Analysis and Exposure Measurement, Vol. 9; Passive Smoking, IARC Publications No. 81, Lyon, France, at 11-23.

[4/] See generally McGill Symposium, supra.

[5/] Baker and Proctor, supra.

[6/] Nystrom, C. and Green, C. (1986). Assessing the impact of environmental tobacco smoke on indoor air quality, Proc. ASHRAE Conf. IAQ 80, Managing indoor air for health and energy conversion, Atlanta, GA, at 213-233.

2024695653

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

2. ETS is not the same as sidestream smoke.[7/] Sidestream smoke is formed at the relatively low temperatures at which a cigarette smolders (around 600 degrees centigrade), and it is carried away vertically from the cigarette in a buoyancy effect.[8/] Less than a second after formation of sidestream smoke the semi-volatile chemical constituents begin to transfer from their initial particulate phase to the vapor phase as the smoke plume is highly diluted and cooled by the surrounding air. As soon as the sidestream smoke is formed, it begins to decay -- with different constituents decaying at different rates.

3. ETS is not the same as mainstream smoke.[9/] Unlike mainstream smoke inhaled by the smoker, ETS is progressively diluted in indoor air until it either is expelled by ventilation or adsorbed on surfaces. This does not mean, however, that ETS may be properly described simply as mainstream smoke in diluted form. Mainstream smoke is a relatively concentrated column of vapor and particulates that is produced at high combustion temperatures (around 900 degrees centigrade)[10/] and is formed in the high humidity environment of the smoker's mouth. Sidestream smoke is produced at lower combustion temperatures and formed in the

---

[7/] Ibid.

[8/] Baker and Proctor, supra.

[9/] Gori and Mantel, supra.

[10/] Baker and Proctor, supra.

2024695654

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

lower humidity environment of indoor air, which results in quite different particulate characteristics.[11/] Mainstream particles are relatively large, moist particles containing many semi-volatile materials. Because of this fundamental distinction, extrapolation of the results of active smoking studies to assess health risks from ETS lacks a valid scientific basis.[12/]

4. In realistic concentrations, ETS is so diluted by indoor air that the most sophisticated analytical equipment cannot identify more than a few of its constituents.[13/] These constituents, in very large part, are no different than those generated by the combustion of any other natural organic material (such as oil).[14/] Accordingly, with few exceptions, the same spectrum of substances will be found in most indoor environments regardless of whether ETS is present. Moreover, the introduction of realistic levels of ETS to most indoor environments adds so little to background levels of combustion

---

[11/] Ingebrethsen, B. and Sears, S. (1985). Particle size distribution measurements of sidestream cigarette smoke, Presented at the 39th Tobacco Chemists Research Conference, Montreal.

[12/] Reasor, M. and Will, J. (1991). Assessing exposure to environmental tobacco smoke: Is it valid to extrapolate from active smoking?, J. Smoking-Related Dis., 2: 111-127; Gori and Mantel, supra.

[13/] Eatough, D., et al. McGill Symposium at 3-39.

[14/] Jenkins, R. and Guerin, M. (1984). Analytical chemical methods for the detection of environmental tobacco smoke constituents, Env. J. Resp. Dis. Suppl., 133: 33-46.

2024695655

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

compounds that little or no increase in such levels can be detected analytically for the majority of substances.[15/]

5. ETS levels can be represented by measurements of airborne nicotine and tobacco-specific respirable suspended particulates. Gross measures of respirable suspended particulates (RSP) are not adequate to identify ETS because there are a variety of indoor air sources of RSP.[16/]

6. ETS is removed from indoor air by ventilation. Ventilation rate is an important factor in the removal rate of ETS from air. Other decay processes are evident, such as deposition and sedimentation, but ventilation is the most significant factor.[17/]

B. ETS Levels in the Workplace

Any evaluation of potential disease risk requires an appropriate exposure assessment.[18/] Adequate data do not exist to establish workplace ETS exposure levels; nevertheless, the bulk of the available research indicates that ETS levels in typical workplace environments, under most

---

[15/] Proctor, C.J. (1988). The analysis of the contribution of ETS to indoor air, Environ. Tech. Lett., 9: 553-562.

[16/] Samet, J., et al. (1987). Health effects and sources of indoor air pollution, Part I, Am. Rev. Respir. Dis., 136: 1486-1508.

[17/] Baker, R., et al. (1988). The build-up and decay of ETS constituents as a function of room conditions, Indoor Air and Ambient Air Quality, Perry, R. and Kirk, P. (eds.), London, Salper, at 121-130. [hereinafter cited as "Perry and Kirk"]

[18/] Human Exposure Assessment of Airborne Pollutants: Advances and Opportunities (1991), National Research Council, National Academy of Sciences, Washington, D.C.

2024695656

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

conditions, generally are much lower than the levels found in home environments.[19/]

A variety of scientific studies have reported levels of airborne nicotine in offices. Nicotine has been used by some as an ETS marker because in air it is one of the few compounds specific to ETS. It also can be found at concentrations that are measurable using modern analytical techniques. These studies typically show levels of airborne nicotine on the order of 5 ug/m3 in smoking areas.[20/] At this level, nicotine concentrations will be somewhat lower than concentrations of most of the volatile organic compounds found in nonsmokers' offices. Because nicotine is one of the most abundant semi-volatile species found in ETS, it is hardly surprising that researchers have noted little difference in the VOC content in the air in smokers' and nonsmokers' offices.[21/] This also explains why so few components of ETS

[19/] Proctor, C., et al. (1991). Assessment of exposure to environmental tobacco smoke in non-smoking British women. Environ. Int., 17: 287-297; Haley, N., et al. (1990). Assessment of environmental tobacco smoke update by non-smokers, Presented at the Society of Behavioral Medicine Annual Scientific Seminar, San Francisco, CA.

[20/] Oldaker, G. III (1990). Results from surveys of environmental tobacco smoke in offices and restaurants, Indoor Air Quality, Kasuga, M. (ed.), Springer Verlag, Berlin, at 99-104 [hereinafter cited as Indoor Air Quality]; Proctor, C., et al. (1989). Measurement of environmental tobacco smoke in an air-conditioned office building, Environ. Tech. Lett., 10: 333-338.

[21/] Baker and Proctor, supra; Jenkins and Guerin, supra; Bayer, C. and Black, M. (1987). Thermal desorption/gas chromatographic/mass spectrometic analysis of volatile organic

(continued...)

2024695657

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

have been measured in office environments -- levels are generally too low and most components non-specific.

Other office studies have measured carbon monoxide, though this gas is not specific to ETS. These studies report that CO levels in offices where smoking occurs are only slightly higher than CO levels in offices where no smoking occurs and that this contribution is only a very small portion of the CO levels typically found in offices.[22/]

Measurement of respirable suspended particulates (RSPs) is another way in which scientists have attempted to quantify ETS exposure levels. As with CO, there are a variety of RSP sources in offices and thus, RSPs lack specificity to tobacco smoke. Some analytical methods, however, do attempt to apportion the contribution of ETS to the particulate levels. These studies have suggested higher RSP levels on the order of 20 to 50% in smokers' offices, with concentrations seldom exceeding 30 ug/m3.[23/]

Overall, the available data suggest that exposure to ETS in offices with smokers is low and that the contribution of ETS to overall indoor air quality is small. That little data on actual personal ETS exposures exist was pointed out in

---

[21/](...continued)
compounds in the offices of smokers and non-smokers, Biomed. and Environ. Mass Spec., 14(8): 363-367.

[22/] Oldaker, supra; Sterling, T., et al. (1987). Environmental tobacco smoke and indoor air quality in modern office work environments, J. Occup. Med., 29: 57-62.

[23/] Oldaker, supra; Proctor, supra.

2024695658

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

a 1988 report by Meridian Research, an organization commissioned by OSHA to review several of the leading reports on ETS after two public interest groups filed a petition asking the agency to prohibit workplace smoking.[24/] In its report, Meridian noted that "there is a paucity of personal sampling data to provide information on the total dose received by a person exposed to passive smoke * * * at work * * * ."[25/] Such data are necessary before exposures to ETS in the workplace realistically and adequately can be quantified.

C. The Alleged Health Effects of ETS

1. Lung Cancer

a. Epidemiologic Studies

Despite the fact that the majority of the epidemiologic studies on ETS and lung cancer have not focused on the workplace, in June 1991 the National Institute for Occupational Safety and Health ("NIOSH") issued a Current Intelligence Bulletin ("CIB")[26/] on "Environmental Tobacco Smoke in the Workplace: Lung Cancer and Other Health Effects,"

[24/] Meridian Research (1988). Review of four studies estimating risk of exposure to environmental tobacco smoke, Report prepared for the Office of Health Standards Programs, Occupational Safety and Health Administration. [hereinafter cited as Meridian Report]

[25/] Id. at 14.

[26/] NIOSH (1991). Environmental tobacco smoke in the workplace: Lung cancer and other health effects, Current Intelligence Bulletin 54. [hereinafter cited as NIOSH CIB]

2024695659

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

in which it asserted that "[r]ecent epidemiologic studies support and reinforce the conclusion [] * * * that exposure to ETS [in the workplace] can cause lung cancer."

Even assuming that the non-workplace epidemiologic studies somehow are relevant to assessing whether ETS is associated with increased risk of lung cancer in the workplace (an assumption that has little scientific validity because of the nature of specific confounding factors that can occur in spousal smoke epidemiologic studies), the NIOSH assertion is questionable. Over the past decade 33 epidemiologic studies, relying on nonsmoker marriage to a smoker as a surrogate for ETS exposure, have been completed worldwide in an effort to assess whether exposure to ETS is associated with an increased risk of lung cancer in the nonsmoker. After adjusting the conclusions to account for misclassification of smokers as nonsmokers, only four of the studies (Geng, Lam I, Lam II, Trichopolous)[27/] -- none of which were conducted in the United States -- found a statistically significant association between nonsmoker marriage to a smoking spouse and lung

---

[27/] Geng, G., et al. (1988). On the relationship between smoking and female lung cancer, Smoking and Health 1987, Aoki, M. and Tominaga, S. (eds.), Amsterdam, Excerta Medica; Lam, T. and Cheng, K. (1988). Passive smoking as a risk factor in never smoking women in Hong Kong, Smoking and Health 1987, ibid.; Lam, T., et al. (1987). Smoking, passive smoking and histological types in lung cancer in Hong Kong Chinese women, Brit. J. Cancer, 56(5): 673-678; Trichopoulos, et al. (1981). Lung cancer and passive smoking, Int. J. Cancer, 27(1): 1-4.

2024695660

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

cancer.[28] All four of the statistically significant studies, moreover, reported only a very weak association, which casts doubt upon their accuracy because of the inherent imprecision of the epidemiologic method when dealing with relative risks of less than 3.0.[29] It also is notable that the more recent studies, such as those published by Wu-Williams, et al.,[30] and Liu, et al.,[31] have taken into account potential confounding factors such as diet, whereas most of the earlier studies did not. These more recent studies show no statistical association between spousal smoke exposure and lung cancer.

Although a study recently published by Fontham, et al.[32] does report a statistically significant association between spousal smoke exposure and adenocarcinoma of the lung, the reliability of these data is questionable. The study reports only the results of the first three years of a five

---

[28] Fleiss, J. and Gross, A. (1991). Meta-analysis in epidemiology, with special reference to studies of the association between exposure to environmental tobacco smoke and lung cancer: A critique, J. Clin. Epidemiol., 44: 127-139.

[29] Lee, P. (1991). Weaknesses in recent risk assessments of environmental tobacco smoke, Environ. Tech., 12: 193-208.

[30] Wu-Williams, et al. (1991). Lung cancer among women in northeast China, Br. J. Cancer, 62: 982-987.

[31] Lui, Z., et al. (1991). Smoking and other risk factors for lung cancer in Xuanwei, China, Int. J. Epidemiol., 20: 26-31.

[32] Fontham, et al. (1991). Lung cancer in non-smoking women: A multi-center case-control study, Cancer Epidemiology, Biomarkers and Prevention, I: 35-45.

2024695661

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

year study and does not find a statistical association for all lung cancers. It was published before diagnoses were verified and urinary cotinine analyses (for assessment of current smoking habit) were completed. Most critically, although the researchers used a questionnaire that acquired data on a range of dietary and lifestyle factors for lung cancer, the paper admits that none of these potential confounding factors were taken into account. Until these issues are addressed, the results of this study are too uncertain to be taken into consideration.

When epidemiologic evidence of an association is weak, scientists agree that it is particularly important that the studies establish a clear dose-response relationship in order to show causality. "Dose-response" is a tendency for the incidence of a disease among individuals exposed to a particular substance to rise as exposure increases. The lack of such a relationship makes it more likely that an apparent association is attributable to the effects of bias or confounding factors and not to exposure. Significantly, evidence of dose-response in the ETS spousal smoking studies is extremely weak, and the U.S. studies either show no dose-response or report inconsistent and contradictory results.[33/]

U.S. cancer trends also are inconsistent with the notion that ETS is a cause of lung cancer in nonsmokers. If it were true that ETS exposure is responsible for more than

---

[33/] Fleiss and Gross, supra.

2024695662

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

one-fourth of lung cancers in U.S. nonsmoking women, a figure derived from the ETS epidemiologic studies, then yearly lung cancer incidence rates among women should have tracked the lung cancer rates among male active smokers over the past 40 years. Lung cancer rates among nonsmoking women, however, have reflected no such trend,[34/] which makes the biological plausibility of an ETS related risk of lung cancer even more questionable.

Any consideration of the potential lung cancer risk from exposure to ETS in the workplace must give priority to studies that have investigated workplace exposure. The key reasons for this are that ETS exposure levels are different in the workplace and the home and epidemiologic studies of spousal ETS exposure are subject to quite different confounding factors (such as the concordance of lifestyle factors between smokers and their wives) than are studies of workplace ETS exposure.

Twelve epidemiologic studies have investigated a possible association between workplace exposure to ETS and lung cancer. One of these studies, reported by Fontham et al.,[35/] is incomplete as discussed above, and hence the association reported in that study should be treated with caution.

[34/] Wynder, E. and Kabat, G. (1990). Environmental tobacco smoke and lung cancer: A critical assessment, Indoor Air Quality at 5-15; Garfinkel, L. and Silverberg, E. (1990). Lung cancer and smoking trends in the U.S. over the past 25 years. Ann. N.Y. Acad. Sci., 609: 46-158.

[35/] Fontham et al., supra.

2024695663

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

Of the other eleven studies, ten report no statistically significant association between workplace exposure to ETS and lung cancer. In fact, all eleven find no statistically significant association with lung cancer in female subjects. Only Kabat and Wynder[36/] reported a barely significant association between workplace exposure and lung cancer in male subjects. However, these authors noted in their paper that this finding was inconsistent with their data on female subjects.

None of the published studies on workplace exposure to ETS and lung cancer are definitive. Most of the studies investigated relatively few lung cancer cases. Moreover, none of the studies adequately measured exposure to ETS but relied instead upon responses to questionnaires. Measurement of actual exposure to ETS in the workplace is critical to a valid determination of potential lung cancer risk, and as yet these data do not exist. Confounding factors also may play an important role in workplace ETS studies, yet those factors have not been addressed properly in relation to the workplace. For these reasons, and because the workplace ETS studies vary considerably in design and were carried out in different countries with disparate workplace conditions, meta-analysis of the workplace ETS studies is of limited scientific value. Nevertheless, a meta-analysis of the work- place ETS studies, including the Fontham study, yields a summary relative risk of

---

36/ Kabat, G. and Wynder, E. (1984). Lung cancer in non-smokers, Cancer, 53: 1214-1221.

2024695664

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

1.01 (95% C.I. 0.92, 1.11); a clearly nonsignificant association between workplace exposure to ETS and lung cancer. See Table 1. Thus, with or without meta-analysis, the available epidemiologic data do not support an association between occupational exposure to ETS and lung cancer.

Although NIOSH purported to address workplace exposure in its CIB, it explicitly acknowledged that the data in the studies it relied upon "were not gathered in an occupational setting."[37/] For reasons it did not explain, NIOSH chose simply to ignore the available epidemiologic studies focusing upon ETS exposure in the workplace. NIOSH also failed to take into consideration studies quantifying levels of ETS in office environments, citing only fragmentary data produced by Repace and Lowrey that is both out of date and in conflict with the other available data.

Instead, NIOSH relies entirely on spousal smoking studies without reconciling their weaknesses and without providing a reliable link between occupational and spousal exposure. Of these studies, moreover, NIOSH fails to address several important recent spousal ETS studies, including the largest completed case-control study performed to date (the Wu-Williams study). The CIB also quotes with approval quantitative risk estimates produced by Repace and Lowrey even though their calculations have been widely criticized by

---

[37/] NIOSH CIB at v.

2024695665

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

independent scientists and government reviews.[38/] In addition, NIOSH stresses the reported association between active smoking and disease without including any discussion of the significant differences in concentration, chemical composition, physical nature and route of inhalation between ETS and mainstream smoke.

The document is replete with omissions and misstatements of fact. To take only a few examples, the Haley data on cotinine is cited to suggest widespread exposure to ETS, but the CIB fails to mention that the same author reported that exposure in the workplace is significantly lower than in the home. It also fails to mention diet as a

---

[38/] Critical reviews of the Repace and Lowrey risk assessment calling their methodology and conclusions into question were conducted by EPA's Carcinogen Assessment Group prior to publication of their paper. See Anderson, E. (no date). "Repace and Lowrey's Estimate of the Lung Cancer Risk from Passive Smoking"; Gibb, H. (no date). "Repace and Lowrey's Estimate of the Nonsmokers' Lung Cancer Risk from Passive Smoking." In addition, the Repace and Lowrey paper was criticized in a review by the Congressional Office of Technology Assessment, Passive Smoking in the Workplace: Selected Issues, at 21-22 (May 1986). Of the numerous scientific articles criticizing the 1985 Repace and Lowrey paper, as well as their earlier work on ETS, see e.g., Gross, A. (1990). Risk assessment relating to environmental tobacco smoke, McGill Symposium at 293-302; Balter, N., et al. (1986). Causal relationship between environmental tobacco smoke and lung cancer in non-smokers: A critical review of the literature, Proceedings of the 79th Annual Meeting of the Air Pollution Control Association; Arundel, A., et al. (1986). Nonsmoker lung cancer risks from tobacco smoke exposure: An evaluation of Repace and Lowrey's phenomenological model, J. of Env. Sci. and Health, 84(1): 93-118; Lebowitz, M. (1986). The potential association of lung cancer with passive smoking, Env. Int., 12: 3-9; Burch, P. (1986). Health risks of passive smoking: Problems of interpretation, Env. Int., 12: 23-28; Kilpatrick, S.J. (1986). Letter to the Editor, Env. Int., 12: 29-31.

2024695666

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

confounding factor, even though it is well documented that women married to smokers generally have a poorer diet than women married to nonsmokers.[39/] NIOSH erroneously asserts that the International Agency for Research on Cancer (IARC) "stated that epidemiologic studies have demonstrated an increased risk of lung cancer for nonsmoking spouses of smokers."[40/] In fact, IARC concluded that the ETS epidemiology is consistent with both an increased risk and no increase in risk -- in other words, the epidemiology is inconclusive.[41/] These and other criticisms are discussed in detail in the attached letters from Samuel D. Chilcote to J. Donald Millar and Bryan D. Hardin.

Given these numerous deficiencies, as well as the fact that the CIB is based almost entirely on data from outside the workplace, the CIB provides no scientific support for the proposition that ETS poses a significant risk in the workplace.

Like the CIB, earlier governmental reports rely almost exclusively on studies of nonsmoking women married to smokers. Each report commented, moreover, on the scarcity of

---

[39/] Sidney, S., et al. (1989). Daily intake of carotene in nonsmokers with and without passive smoking at home, Am. J. Epi., 129: 1305-1309; Koo, L. (1988). Dietary habits and lung cancer risk among Chinese females in Hong Kong who never smoke, Nutrition and Cancer, 11: 155-172.

[40/] NIOSH CIB at 18.

[41/] IARC (1986). Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Tobacco Smoking, Vol. 38, at 308.

2024695667

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

reliable workplace data. The Surgeon General's 1986 report, for example, observed that "the workplace * * * was not considered in the early studies on involuntary smoking," and later studies of workplace exposure have been "limited and inconclusive."[42/]

The National Research Council, which reviewed all but one of the same studies relied upon by the Surgeon General, also did not address occupational exposure in its 1986 report. The NRC Report noted that "[m]ost epidemiologic studies of chronic health effects have been conducted on persons who have had a long term exposure to ETS from household members * * * [and] do not directly address chronic health effects in individuals who are exposed at work or have occasional exposures in the home or elsewhere."[43/]

b. Meta-Analysis of ETS Exposure Studies

Any meta-analysis of ETS exposure studies generates no more than a very weak statistical association between ETS and lung cancer. In any epidemiologic study indicating a weak association between exposure to a substance and cancer, potential confounding factors must be seriously considered. At least 20 variables are associated with an increased risk of lung cancer, including diet, occupation, alcohol consumption,

---

[42/] U.S. Surgeon General (1986). The Health Consequences of Involuntary Smoking, U.S. Department of Health and Human Services 91 [hereinafter cited as Surgeon General's Report].

[43/] National Research Council (1986). Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects at 7. [hereinafter cited as NRC Report]

2024695668

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

geographic location and heredity.[44] As discussed in the Meridian Report to OSHA, these variables must be considered and ruled out before an association between ETS and lung cancer can be considered definitive.[45] Indeed, it has been observed in the case of ETS that it may not be the potential health effects of ETS that the scientific community recently has been evaluating but, rather, the relative lung cancer risk of a nonsmoker leading a "smoker's lifestyle" as evidenced by marriage to a smoker.[46]

The approach to meta-analysis often taken by those purporting to find a statistically significant association between spousal exposure to ETS and lung cancer is to ignore the fact that the potential for, and types of, bias and confounders to be ruled out will vary among the individual ETS/lung cancer epidemiologic studies. The Environmental Protection Agency, for example, claimed to take misclassification bias into account in its draft ETS/lung cancer risk assessment document[47] but failed to adjust the studies

---

[44] Rylander, R. (1990). Environmental tobacco smoke and lung cancer, New Eng. J. Med., 323: 834; Gori and Mantel, supra.

[45] Meridian Report, supra.

[46] Koo, L. et al. (1987). Measurements of passive smoking and estimates of lung cancer risk among non-smoking Chinese females, Int. J. Cancer, 39: 162-169.

[47] U.S. Environmental Protection Agency (1990). Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disorders in Children, Review Draft, EPA/600/6-90/006A. [hereinafter cited as EPA Draft Risk Assessment]

2024695669

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

individually on this basis. In effect, this approach assumes erroneously that the percentage of smokers denying smoking in an epidemiologic questionnaire is the same in the U.S. as in Asia and Europe. Additionally, EPA completely neglected to evaluate other potential biases in the studies. Such an approach to meta-analysis is scientifically unacceptable.

Clearly, in many of the ETS studies recall bias potentially is quite large;[48/] precisely how large depends upon the particular methodology employed. Recall of spousal smoking habits has been shown to vary dramatically depending on information source. An American Cancer Society study, for example, reported a relative risk of 0.92 when husbands provided the information on smoking status, a risk of 1.0 when the cases reported the information, and a risk of 3.19 when sons and daughters provided the information.[49/] The unreliability of the last measure is obvious when one considers that many of the children seeking to recall their parents' smoking habits (individuals in their 60s, 70s and 80s) had been away from their parents' homes for many years.

Moreover, because the ETS studies were conducted in a large number of countries with widely varying cultures, the potential for misclassification of smoking status --

---

[48/] Wynder, E. (1987). Guidelines to the epidemiology of weak associations, Prev. Med., 16: 211-212.

[49/] Layard, M. (1990). Environmental tobacco smoke and cancer: The epidemiologic evidence, McGill Symposium at 99-115.

2024695670

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

particularly the smoking status of women -- certainly should be expected to vary among the individual ETS/lung cancer epidemiologic studies. For cultural reasons, a Japanese woman who smokes may be more likely to deny that she does than is an American woman who smokes. Yet many of the meta-analyses of the ETS/lung cancer epidemiologic studies purporting to show a statistically significant association between spousal exposure to ETS and lung cancer -- including EPA's in the draft ETS risk assessment -- fail to account for this by not adjusting each study individually for misclassification.[50/]

Instead, EPA relied almost entirely on misclassification data from the United Kingdom reported by Wald[51/] resulting in an unjustifiably low misclassification rate. Not only did Wald use a very high threshold level for distinguishing smokers from nonsmokers, he also included one study in his combination of four that reported urinary nicotine. This latter study gave a misclassification rate of 0%, not surprising given the very short half-life of nicotine. The subject of the study would have had to have smoked a cigarette almost immediately before the interview in order to

---

[50/] Lee, P. (1988). Misclassification of Smoking Habits and Passive Smoking. A Review of the Evidence, Springer-Verlag, Berlin; Lee, P. (1988). An alternative explanation for the increased risk of lung cancer in non-smokers married to smokers, Perry and Kirk, at 149-158; Schwartz, S. and Balter, N. (1988). ETS-lung cancer epidemiology: Supportability of misclassification of risk assessment, Environ. Tech. Lett., 9(6): 479-490.

[51/] Wald, et al. (1986). Does breathing other people's tobacco smoke cause lung cancer?, Br. Med. J., 293: 1217-22.

2024695671

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

detect a significant level of nicotine. A review of U.S. misclassification rates would suggest a rate of 6-8% rather than the 2% used by EPA.[52/]

In addition, many of the meta-analyses of the ETS/lung cancer studies purporting to show a statistically significant association fail to account for numerous confounding factors, quite a few of which might be expected to vary significantly among studies conducted in different countries all over the world. As indicated previously, the weak purported association between spousal exposure to ETS and lung cancer requires that the possibility of all confounders be examined thoroughly and ruled out. In addition, the potential for certain confounders thought to be related to smoking patterns and to cancer rates such as race, ethnicity and cultural background, surely depends upon the area of the world in which a given study is conducted. Furthermore, other potentially important confounding factors appear to be peculiar to individual countries.[53/] This reinforces the need for critical review and adjustment of each study individually prior to meta-analysis.

[52/] Lee, P. (1987). Passive smoking and lung cancer association: A result of bias?, Human Tox., 6: 517-524.

[53/] See, e.g., Koo, L. (1989). Environmental tobacco smoke and lung cancer: Is it the smoke or the diet?, Bieva, C.J., et al. (eds.), Present and Future of Indoor Air Quality, Excerpta Medica, New York; Koo, L. et al. (1988). Life-history correlates of environmental tobacco smoke: A study of nonsmoking Hong Kong Chinese wives with smoking versus nonsmoking husbands, Soc. Sci. Med., 26: 751-760.

2024695672

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

Finally, the potential impact of publication bias is particularly great when meta-analysis pools published studies to discern a trend. There is strong reason to believe that scientists are less likely to submit,[54] and scientific journals less likely to accept, epidemiologic studies not reporting an association between exposure to a particular substance and disease than those showing a positive association.[55] In the case of ETS, the observation that studies reporting the highest relative risks are often those based on significantly smaller numbers of cases than the studies reporting the lowest relative risks is consistent with the notion that small null studies do not get published.[56] Indeed, it is doubtful that the recently published Fontham study[57] would have been submitted and published in its current incomplete form if the data did not indicate a positive association.

---

[54] Dickersin, K., et al. (1992). Factors influencing publication of research results, JAMA, 267: 374-378.

[55] Gordis, L. (1988). Challenges to epidemiology in the next decade, Am. J. Epi., 128: 1-9; Newcombe, R. (1987). Towards a reduction in publication bias, Brit. Med. J., 295: 656-659.

[56] Lee, P. (1991). Weaknesses in recent risk assessments of environmental tobacco smoke, Environ. Tech. Lett., 12: 193-208, 197; Buffler, P. (1988). The evaluation of negative epidemiologic studies: The importance of all available evidence in risk characterization, Reg. Tox. and Pharmacology, 9: 34-43.

[57] Fontham, supra.

2024695673

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

The EPA Science Advisory Board ("SAB") has recognized many of the shortcomings in the ETS/lung cancer epidemiology. In its 1991 report reviewing EPA's draft risk assessment on ETS,[58] the SAB advised the agency not to rely heavily on the epidemiology because of the weakness of the association purported to be demonstrated by EPA's meta-analysis, the failure of many of the studies adequately to consider confounding factors and the possibility that misclassification of smokers as nonsmokers may have skewed the reported results.[59]

The SAB's suggested alternative approach, -- that the "case for cancer" might better be made by extrapolating the science on active smoking to ETS -- also is invalid. As explained fully in Part A, the suggestion that ETS issues can be resolved by simple extrapolation from active smoking data is both simplistic and not supported by the existing science.

Even if there were a legitimate basis to compare epidemiologic studies of active smoking with studies of

---

[58] EPA Science Advisory Board (1991). Review of the EPA's Office of Research and Development Document: Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disorders in Children, EPA/600/6-90/006A, June 1990.

[59] IARC has observed, for example, that ETS epidemiologic studies have encountered "substantial difficulties in determination of passive exposure to tobacco smoke and to other possible risk factors for the various cancers studied. The resulting errors could arguably have artefactually depressed or raised estimated risks, and, as a consequence, each is compatible either with an increase or with an absence of risk." IARC Monograph at 308.

2024695674

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

spousal smoking, the comparison would not support a finding of significant risk from exposure to ETS. Consistent epidemiologic data indicate that active smoking of less than 4 cigarettes per day is not associated with a significantly increased risk of lung cancer.[60/] Because average doses of ETS exposure are several thousand times less than exposure levels from active smoking, a significant lung cancer risk from ETS exposure would not be expected on the basis of the active smoking data, and the marginal relative risks reported in some ETS epidemiologic studies are likely to be statistical artifacts, derived from unaccounted confounders and unavoidable bias.[61/]

Even adopting the method and assumptions used by EPA in preparing its ETS draft risk assessment, moreover, a meta-analysis of worldwide ETS epidemiological studies that includes the most recent studies shows no statistically significant association. See Table 2. A meta-analysis of the United States studies also shows no significant association. See Table 3. Even the ETS draft risk assessment acknowledges that EPA's meta-analysis of the raw data from the U.S. studies

---

[60/] Gori and Mantel, supra; Doll, R. and Peto, R. (1978). Cigarette smoking and bronchial carcinomas: Dose and time relationships among regular smokers and lifelong non-smokers, J. Epidemiol. Commun. Health, 32: 303-315.

[61/] Gori and Mantel, supra.

2024695675

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

shows no such association.[62/] The NRC reported similar results from its own meta-analysis of the U.S. studies.[63/]

In sum, the available epidemiologic evidence does not convincingly support a finding that exposure to ETS is associated with an increased risk of lung cancer among nonsmokers in general, nor does reliable evidence support a conclusion that exposure to ETS in the workplace is associated with an increased risk of lung cancer. The epidemiologic studies focusing on workplace ETS exposure do not support the suggestion that ETS increases the risk of lung cancer. Moreover, workplace ETS exposure levels have not been adequately quantified, and therefore no basis exists for extrapolating from spousal ETS studies or concluding that ETS poses a significant risk of lung cancer in the workplace.

2. Cardiovascular Disease

In 1986, following a review of the four epidemiologic studies that were available at that time, the Surgeon General concluded that "[f]urther studies on the relationship between involuntary smoking and cardiovascular disease are needed in order to determine whether involuntary smoking increases the risk of cardiovascular disease."[64/] A 1991

[62/] EPA Draft Risk Assessment at 4-39.

[63/] NRC Report at 231.

[64/] Surgeon General's Report at 107-08.

2024695676

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

review article by Glantz and Parmley[65/] claiming that ETS might be responsible for as many as 37,000 heart disease deaths per year in the United States suggests that this situation has changed. Such a claim is not adequately supported by the scientific data.

To date, 11 epidemiologic studies (including the four considered by the Surgeon General in 1986) have been conducted in an effort to determine whether exposure to ETS leads to an increased risk of cardiovascular disease in the nonsmoker. These 11 studies have reported 13 different estimates of relative risk, with some studies reporting only for women, others reporting only for men, some reporting for both sexes together and still others reporting separately for men and women. See Table 4. Of the 13 relative risks reported, only four are statistically significant, and these four have been criticized by commentators for failing to control for many of the almost 300 cardiovascular disease risk factors identified to date.[66/] It also has been suggested

---

[65/] Glantz, S. and Parmley, W. (1991). Passive smoking and heart disease, Circulation, 83: 1-12.

[66/] Armitage, A.K. (1991). Environmental tobacco smoke and coronary heart disease, Other People's Tobacco Smoke, A.K. Armitage (ed.), Yorkshire, U.K., Galen Press, at 109-116; Weetman, D.F. and Munby, J. (1990). Environmental tobacco smoke (ETS) and cardiovascular disease, Indoor Air Quality and Ventilation, Lunau, F. and Reynolds, G.L. (eds.), London, Selper Ltd., at 211-216; Thiery, J. and Cremer, P. (1990). Coronary heart disease and involuntary smoking, Presented at the Toxicology Forum, Session on Environmental Tobacco Smoke: Science and Meta-Science, Budapest, Hungary; Wexler, L. (1990). Environmental tobacco smoke and cardiovascular

(continued...)

2024695672

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

that misclassification of smokers as nonsmokers may account for the results reached in some of these studies. Only one of these studies attempted to measure exposure in the workplace though other studies do mention the workplace without providing substantial data. The one study with workplace specific data reported no statistically significant association, with relative risks of 0.95 for males and 0.66 for females.[67/]

Finally, and most significantly, the results of these studies are not biologically plausible given the relatively weak association reported between active smoking and cardiovascular disease. A relative risk of around 2 has been reported for active smoking and cardiovascular disease, with a somewhat lower relative risk for women than men. Glantz and Parmley claim a relative risk for spousal smoke exposure and cardiovascular disease in women of 1.3 -- a risk that is virtually equivalent to that found for active smoking. Because the difference in exposure to mainstream smoke and ETS is of the order of a thousand-fold, the closeness of the two

---

[66/](...continued)
disease, McGill Symposium, at 139-152; NRC Report at 263-65; Holcomb, L. (1991). Environmental tobacco smoke and cardiovascular disease, Circulation, 84: 957-58.

[67/] Dobson, et al. (1991). Passive smoking and the risk of heart attack or coronary death, Med. J. Australia, 154: 793-97.

2024695678

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

claimed relative risks is implausible. This apparent inconsistency was acknowledged by NIOSH in their CIB.[68/]

Almost exactly one year after Glantz and Parmley published their much-criticized article on ETS and heart disease, Steenland has published a review article[69/] reaching similar conclusions. The author, a NIOSH researcher, claims that heart disease from ETS exposure likely is a greater public health threat than lung cancer and estimates that 35,000 to 40,000 heart disease deaths per year during the early 1980s occurred in never-smokers and ex-smokers as a result of ETS exposure. He also asserts that experimental studies have shown that ETS can damage the cardiovascular system through both short- and long-term mechanisms, suggesting that childhood exposure may play a role in cardiovascular disease.

Steenland's conclusions are questionable for many of the same reasons as Glantz's and Parmley's. He fails to account for the lack of biological plausibility for an association between ETS and heart disease and relies, in part, upon the same four studies reviewed by the Surgeon General in 1986 and determined not to establish that ETS exposure can lead to heart disease. He attaches a great deal of weight to

---

[68/] NIOSH CIB at 11.

[69/] Steenland, K. (1992). Passive Smoking and the Risk of Heart Disease, J. Amer. Med. Ass., 267: 1.

2024695679

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

the Helsing study,[20] which is severely flawed in that it failed to collect data even on the most well-known risk factors for coronary heart disease, including diet, exercise, blood pressure and cholesterol levels. The experimental studies that Steenland describes as supporting the epidemiologic evidence involved exposing subjects to levels of carbon monoxide many times above conceivable levels to be encountered in any workplace. Steenland's assertions also depend entirely upon the assumption that the epidemiology reports a relationship between ETS exposure and cardiovascular disease, an association that Steenland himself admits is uncertain.

Finally, his estimate of annual deaths from heart disease is not only invalid but misleading. Steenland observes that "[d]ifferential misclassification of ever-smokers as never-smokers and uncontrolled confounding are possible explanations for the excess risk observed in the epidemiologic studies."[21] In other words, whether any risk of coronary heart disease from ETS exposure exists at all remains uncertain.

Research also has called into question the biological plausibility of claims that ETS causes acute effects in individuals with pre-existing coronary artery

---

[20] Helsing, K. et al. (1988). Heart disease mortality in nonsmokers living with smokers, Am. J. Epidemiol., 127: 915-922.

[21] Steenland, supra, at 98.

2024695680

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

disease. In one study,[72] conducted after the authors of an earlier study suggested that ETS exposure elevates carboxyhemoglobin levels in the nonsmoker to the point of triggering acute angina,[73] thirty individuals with well-documented coronary artery disease and symptomatic angina were exposed in a chamber to carbon monoxide, using an endpoint of approximately four percent carboxyhemoglobin. To reach this exposure level, the researchers were forced to expose the subjects to 100 ppm of carbon monoxide -- around a hundred times higher than the level that might be expected to be contributed by ETS in an office environment -- but they identified no effect of the ETS exposure on the onset of angina.

In short, claims that ETS exposure leads to an increased risk of cardiovascular disease or can trigger acute coronary effects at realistic exposure levels are not supported by convincing scientific data. In addition, there are few data that suggest that ETS exposure in the workplace has any relation to cardiovascular disease.

---

[72] Sheps, D., et al. (1987). Lack of effect of low levels of carboxyhemoglobin on cardiovascular function in patients with ischemic heart disease, Arch. Environ. Health, 42: 108-116.

[73] Aronow, W. (1978). Effects of passive smoking on angina pectoris, New Eng. J. Med., 299: 21-24. The Aronow study has been widely criticized for a variety of flaws, including the use of subjective pain as an end point without double blinding, a very small sample size, the tendency of patients to produce symptoms suggested to them, and failure to control for stress. Wexler. McGill Symposium at 149.

2024695681

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

3. Respiratory Effects on Healthy Adults

Few studies have been published during the past five years on the effects of ETS on adult respiratory health. The reason, at least in part, is that this issue was largely settled by 1986 when the ETS reports of the Surgeon General and National Academy of Sciences were published.

According to the Surgeon General, the studies available in 1986 indicated "that a previously healthy individual would not develop chronic lung disease solely on the basis of * * * [exposure to ETS in adult life]."[74/] Similarly, the Surgeon General found that, even when confronted with artificially high doses of ETS in clinical tests, test subjects experienced at most "quite small" changes in air flow from the lung. This led the Surgeon General to conclude that "it is unlikely that this change in airflow, per se, results in symptoms."[75/] The NRC Report reached similar conclusions.[76/]

Studies of ETS and respiratory health in normal, healthy adults that have been published since 1986 have generally reached similar conclusions to those of the earlier studies.[77/] Of these more recent studies, one by Kentner, et

---

[74/] Surgeon General's Report at 62.

[75/] Surgeon General's Report at 63.

[76/] NRC Report at 199-200.

[77/] Witorsch, P. (1990). Effects of ETS exposure on pulmonary function and respiratory health in adults, McGill Symposium, at 169-186.

2024695682

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

al. focuses specifically on workplace exposure to ETS and pulmonary function.[78] The study examined 1,364 employees with healthy lungs from three different companies over a two-year period and reports that "everyday passive exposure to smoke in the office or at home does not cause a significant impairment of lung function in adults with healthy lungs."[79] Thus, from this study there is little evidence to suggest that ETS adversely affects lung function or respiratory health in healthy adults. There are few other studies pertaining to occupational ETS exposure and respiratory problems. The studies that have been conducted on this issue have reported contradictory results.[80]

4. Respiratory Effects on Sensitive Individuals

Several groups of investigators have focused on whether ETS presents problems for people already suffering from asthma or claiming a special sensitivity to tobacco smoke. A clinical approach has been employed in these investigations, with case and control subjects being placed in enclosures and exposed to machine-generated ETS -- often at levels substantially exceeding real-life exposure levels by

---

[78] Kentner, M., Weltle, D. and Valentin, H. (1989). Passive smoking and lung function in adults, Arbeitsmedicine Sozialmedicine Praventivmedicine, 24: 8-13.

[79] Ibid.

[80] White, J., et al. (1991). Respiratory illness in nonsmokers chronically exposed to tobacco smoke in the workplace, Chest, 100: 39-43; Kentner, M. and Weltle, D. (1985). Passive tobacco smoke inhalation and lung function in adults, Perry and Kirk, at 232-241.

2024695683

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

orders of magnitude. The results of these tests (with concentrations such that CO levels were 20-30 ppm) have been inconclusive. In fact, even at unrealistically high exposure levels, measurable responses to ETS have been difficult to elicit.[81/]

One group of investigators has suggested that a small subset of asthmatics may be intolerant to ETS.[82/] The same investigators have failed to find, however, any relationship between perceived intolerance, as determined by pulmonary function testing or the subject's own statements, and sensitivity or reaction to tobacco leaf extract. This finding is consistent with earlier reports finding no association between subjectively reported sensitivity to ETS -- by some asthmatics as well as others claiming to be smoke-sensitive -- and objective immunologic tests. Additionally, recent research has suggested that hypersensitivity to ETS may be psychosomatic.[83/]

One difficulty that scientists have faced in this area is the fact that asthmatic episodes can be triggered by stress as well as by environmental factors. Identifying the actual cause of an asthmatic episode from a long list of

---

[81/] Witorsch. McGill Symposium at 178.

[82/] Stankus, R., et al. (1988). Cigarette smoke-sensitive asthma: Challenge studies, J. Allergy Clin. Immun., 82: 331-338.

[83/] Weidemann, H., et al. (1986). Acute effects of passive smoking on lung function and airway reactivity in asthma subjects, Chest, 89: 180-195.

2024695684

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

candidate substances is, moreover, seldom easy. As the Surgeon General observed in 1986,[84] "biases in observation and in the selection of subjects and the subjects' own expectations may account for the widely divergent results" that have been reported in studies of ETS and asthmatics. The NRC also noted that "pulmonary function changes [in asthmatic subjects] could be emotionally related to cigarette smoke exposure."[85]

5. ETS and Sick Building Syndrome

Because ETS is the most visible component of indoor air, many people wrongly attribute complaints about sore eyes, dry throats and other symptoms of poor indoor air quality to it. The coincidence of smoking employees and an outbreak of symptoms is simply assumed to be causally related.

In fact, substantial accumulations of tobacco smoke are a visible symptom of an underlying problem in the ventilation of an indoor environment, not a cause. In a study of 203 "sick" buildings from 1978 to 1983,[86] for example, NIOSH found that only four had indoor air quality problems attributable to ETS but that approximately one-half were inadequately ventilated. Other researchers have reported

---

[84] Surgeon General's Report at 65.

[85] NRC Report at 215.

[86] Melius, J., Wallingford, K., Keenlyside, R., and Carpenter, J. (1985). Indoor Air Quality -- The NIOSH Experience, Presentation to the American Society of Heating, Refrigeration & Air Conditioning Engineers, Atlanta, GA.

2024695685

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

similar findings.[87/] In those few cases where high concentrations of ETS have been found in "sick" buildings (by NIOSH and others), moreover, other indoor air contaminants also have been found at excessive levels. In most cases, therefore, tobacco smoke, because of its visibility, is merely a scapegoat for inadequate ventilation.

Poor ventilation allows airborne substances to accumulate inside a building. Studies show that symptoms often attributed to ETS may be the result of exposure to contaminants such as formaldehyde, carbon monoxide, oxides of nitrogen, ozone, fungal and bacterial spores, cotton fibers and fiberglass fragments. These pollutants, coupled with poorly maintained and inadequate ventilation systems, are the real causes of sick building syndrome.

The terms "sick building syndrome" and "building related illness" are relatively new. For the most part, they are a function of two modern developments: the rise of centrally air-conditioned buildings with sealed windows and the energy crisis of the 1970s. Conservation efforts have led many building owners and operators to reduce the amount of fresh air entering their buildings. Often, stale, dirty air

---

[87/] Liao, S.S.T., Bacon-Shone, J. and Kim, Y.S. (1991). Factors influencing indoor air quality in Hong Kong: Measurements in offices and shops, Environ. Tech., 12: 737-745; Hedge, A., Erickson, W.A. and Rubin, G. (1991). The effects of smoking policy on indoor air quality and sick building syndrome in 19 air-conditioned offices. Presented at IAQ '91, Healthy Buildings, Washington, D.C.; Robertson, G. (1990). Indoor pollution: Sources, effects and mitigation strategies, McGill Symposium, at 333.

2024695686

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

is simply recycled continuously through the building. Air quality is further compromised by inadequate filtration and contamination of dirty ductwork by bacteria, molds and fungi. In these circumstances, it is not surprising that building occupants may be afflicted by a variety of physical ailments.

Building owners and managers sometimes have responded to occupant complaints by imposing total smoking bans in their buildings. Since ETS is a factor in only a small percentage of cases, however, smoking bans will not solve the problem. For example, one researcher reports the case of a new office building in the Washington, D.C. area that became "sick" shortly after the employees moved in.[88/] In response to rising employee complaints and increased absenteeism, management called in a health inspector. Observing smoke-filled offices, he recommended a smoking ban, which failed to resolve the problem. A more comprehensive investigation revealed major ventilating system problems, including sealed outdoor air intakes, cheap, inadequate filters, and microbial contamination in the air handling units. Improved ventilation and maintenance resolved the problem, and no smoking restrictions were necessary.

Perhaps the most celebrated example of such a building is the EPA's own headquarters building in Washington, D.C. According to press accounts, many EPA employees became

---

[88/] Turner, S. (1991). Sick buildings: Causes and effects, Presented at the Symposium on Indoor Air Quality and Health, Universita Degli Studi di Perugia, Perugia, Italia.

2024695687

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

so ill that EPA was forced to move their offices to a nearby apartment building. An investigation revealed that the problem was caused primarily by chemicals emitted from newly installed carpeting, not by ETS.[89/]

6. Irritating Effects

If ETS is permitted to accumulate, it may become an annoyance -- to both smokers and nonsmokers. So can many other substances found indoors, including formaldehyde from carpets and wallboards and ozone from office copying machines. In fact, literally hundreds of chemicals can reach excessive levels indoors from myriad sources and activities if ventilation is inadequate and ventilation system filtration is deficient.

The importance of proper ventilation in addressing indoor air quality problems comprehensively is reflected in the national building ventilation standard adopted by the American Society of Heating, Refrigeration and Air Conditioning Engineers (ASHRAE) and approved by the American National Standards Institute (ANSI). ANSI/ASHRAE Standard 62-1989[90/] recommends a ventilation rate of 20 cubic feet per minute of fresh air for each person (with 15 cfm as the minimum) as adequate to deal with typical smoking levels as

---

[89/] Weiskopf, M. "For EPA, War on Pollution Strikes Home," The Washington Post, Dec. 12, 1989 at A23

[90/] American Society of Heating, Refrigeration and Air-Conditioning Engineers (1989). Standard 62-1989, Ventilation for Acceptable Indoor Air Quality.

2024695688

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

well as most other building contaminants. The 15 cfm/person minimum is intended to ensure that the carbon dioxide level in occupied spaces does not exceed 1,000 parts per million over long-term use. This level of carbon dioxide is consistent with actions that have been taken by other countries, such as Japan (1,000 ppm). The standard also describes acceptable concentrations of various indoor contaminants, which is an alternative to the ventilation-based approach. If OSHA concludes that regulation of indoor air quality is necessary, a requirement that employers ventilate to ANSI/ASHRAE Standard 62-1989 is the single most effective step it could take to safeguard the comfort and health of employees.

II. ETS EXPOSURE IN THE WORKPLACE IS NOT ASSOCIATED WITH A "SIGNIFICANT RISK" OF MATERIAL IMPAIRMENT OF HEALTH

A. The Legal Standard

As a prerequisite to regulation of workplace exposure to a particular substance, OSHA must prove that the regulation is necessary to remedy a significant risk of a material health impairment. Based on the foregoing, it is clear that there is an inadequate factual basis for OSHA to regulate workplace exposure to ETS.

The Occupational Safety and Health Act of 1970 provides that the Secretary of Labor is authorized "to set mandatory occupational safety and health standards * * * ." 29 U.S.C. § 651(b)(3) (1988). Section 3(8) of the Act defines an "occupational safety and health standard" as a standard which "requires conditions, or the adoption or use of one or

2024695689

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

more practices, means, methods, operations, or processes, reasonably necessary or appropriate to provide safe or healthful employment and places of employment." 29 U.S.C. § 652(8) (1988).

The statute imposes additional requirements with respect to "toxic materials or harmful physical agents." Section 6(b)(5) of the Act requires the Secretary to promulgate a "standard which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity even if such employee has regular exposure to the hazard dealt with by such standard for the period of his working life." 29 U.S.C. § 655(b)(5) (1988).

The Supreme Court has held that in order to promulgate a standard regulating toxic materials or harmful physical agents, OSHA must satisfy the substantive requirements of both § 3(8) and § 6(b)(5). In Industrial Union Department, AFL-CIO v. American Petroleum Institute, 448 U.S. 607 (1980), ("Benzene"), a plurality of the Supreme Court held that § 3(8) must be satisfied before there can be any consideration of a standard under § 6(b)(5). The plurality opinion stated that "[Section 3(8)] requires the Secretary, before issuing any standard, to determine that it is reasonably necessary and [sic] appropriate to remedy a significant risk of material health impairment." Id. at 639. In effect, the plurality opinion interpreted the definitional

2024695690

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

provisions of section 3(8) to operate as a substantive limitation on OSHA's rulemaking authority. Id. at 690 (Marshall, J., dissenting). Thus, § 3(8) imposes a dual requirement on the agency. First, the standard must be intended to remedy a "significant risk of material health impairment" and, second, it must be "reasonably necessary or appropriate" to achieve this purpose. In imposing the significant risk requirement, moreover, the Court expressly rejected OSHA's preexisting policy of regulating carcinogens to the "lowest feasible level."

The Court stressed that, in regulating a purported carcinogenic substance, OSHA must not only prove that the substance causes cancer at some level of exposure but must also suffuciently quantify the risk to workers to demonstrate in an understandable way that workplace exposure levels pose a significant risk. In setting a workplace exposure limit, OSHA cannot rely exclusively upon adverse effects observed at exposure levels or under circumstances that have not been shown to have a valid relationship to occupational exposure.

As with any substantive requirement imposed by the Act, a finding of "significant risk" must be supported by substantial evidence in the record considered as a whole. The substantial evidence standard requires more rigorous scrutiny of an agency's decision than the "arbitrary and capricious" test generally applicable to informal rulemaking. AFL-CIO v. Marshall, 617 F.2d 636, 649 (D.C. Cir. 1979) ("Marshall")

2024695691

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

(footnote omitted). Congress adopted this more stringent standard of review in order "to provide a careful check on the agency's determinations without substituting its judgment for that of the agency" and "to check extravagant exercises of the agency's authority to regulate risk." Id. at 649-50 (footnote omitted). See also National Grain and Feed Ass'n v. OSHA, 858 F.2d 1019, 1030 (5th Cir. 1988); Asbestos Information Ass'n/ North America v. OSHA, 727 F.2d 415, 421 (5th Cir. 1984).

As OSHA itself has stated in a recent court filing in response to a petition by Action on Smoking and Health (ASH) to ban or restrict workplace smoking, the legal requirement of significant risk "means that each standard must be supported *by an enormous amount of evidence and analysis relating both to worker health, and the feasibility of mandated controls.*" The Secretary's Response to the Court's Order for Further Submissions, Action on Smoking and Health v. OSHA, Nos. 91-1037, 91-1038 at 5 (D.C. Cir., Dec. 20, 1991) ("Secretary's Response")(emphasis added).

B. The Available Evidence Does Not Support a Finding of Significant Risk

This evidentiary burden cannot be satisfied by the available evidence pertaining to workplace exposure to ETS. First, as discussed in Part I, the available epidemiologic evidence does not support the contention that ETS poses a significant risk to the health of nonsmokers. Second, there is very little data on ETS exposure in the workplace and the available epidemiologic data do not support a statistically

2024695692

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

significant association between exposure to ETS in the workplace and lung cancer or other disease. Of the 11 occupational ETS studies, 10 have reported no significant association.

In addition, OSHA's own criteria for the development of risk assessments specify that among the most important components to be considered in performing quantitative risk analyses are (1) a description of the potential exposure and worker scenarios and (2) a description of the dose-response relationship and a quantitative determination of risk. See Occupational Exposure to Inorganic Arsenic; Supplemental Statement of Reasons for the Final Rule, 48 Fed. Reg. 1864, 1867-68 (1983). As discussed below, the available data do not meet these criteria.

1. Inadequate Evidence of Significant Risk

As shown in these comments, there is no reliable evidence that ETS exposure poses a significant risk of lung cancer or other serious disease to any population group. The available epidemiologic studies are weak and inconclusive, and a meta-analysis of the published worldwide ETS epidemiologic studies does not show a statistically significant risk of lung cancer. Moreover, there is no evidence from clinical tests, animal studies or other sources that ETS exposure causes a material health impairment.

2024695693

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

2. Insufficiency of Workplace Exposure Data

Among the information critical to any risk assessment is reliable data on exposure, "including data on the concentration of ambient tobacco smoke components in indoor workplace air." Secretary's Response at 6 (footnote omitted). See also Occupational Exposure to Indoor Air Pollutants; Request for Information, 56 Fed. Reg. 47,892, 47,894-95, Questions 34(a)-39(c) (1991). The meager amount of data that exist in this area would not support a finding of significant risk. As the agency has made clear, "OSHA still does not have adequate information on the current level of exposure to ambient tobacco smoke in workplaces to accurately assess the existing level of occupational risk." Secretary's Response at 6 (citations omitted). We are aware of no studies or reports that would alter this conclusion or remedy the substantial gaps in the data.

An article published just last year by Dr. Wayne Ott confirms this conclusion.[21/] Dr. Ott is the former head of the Air, Toxics and Radiation Research Team of EPA's Office of Modeling, Monitoring Systems and Quality Assurance, which is responsible for developing methods for modeling and measuring pollutants in the environment. Dr. Ott noted that the various exposure assessment and monitoring methods currently available for making ambient measurements "seldom are adequate for

---

[21/] Ott, W. (1990). Total human exposure: Basic concepts, EPA field studies, and future research needs, J. Air Waste Manage. Assoc., 40: 966-975.

2024695694

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

monitoring human exposure." Among the principal research needs identified by Dr. Ott is the "[s]tudy of environmental tobacco smoke in office buildings." In discussing the results of EPA's Total Exposure Assessment Methodology ("TEAM") studies, Dr. Ott also described the many unanswered research questions suggested by these studies, including "[w]hat is the relative importance of home and workplace exposure to environmental tobacco smoke?" Similarly, in its 1989 Report to Congress, EPA identified research on ETS exposure and on dosimetry as a top priority. EPA's own draft ETS risk assessment, moreover, contains no exposure data and no exposure assessment because EPA has yet to make any significant progress in conducting this critical research.

3. Inadequate Dose-Response Information

Even if OSHA were able to establish that ETS constitutes a significant risk at some level of exposure, there is inadequate dose-response information to support an ETS standard. As pointed out in Section I, the evidence for a dose-response relationship is weak and the U.S. studies either show no dose-response or report inconsistent results.

In Benzene, the Supreme Court specifically rejected OSHA's refusal to construct a dose-response curve correlating exposure levels and cancer risk, observing that such an analysis could be important in evaluating whether there is a significant risk. Benzene at 667, n. 64. Since Benzene, a quantitative risk assessment showing the degree of risk to

2024695695

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

workers at various exposure levels therefore has been required in order to establish the existence of a significant risk. See 48 Fed. Reg. at 1867-68.

Benzene also made clear that OSHA cannot rely on evidence of a purported carcinogenic effect at high exposure levels to set lower exposure levels in lieu of reliable dose-response information. "As the discussion of the Agency's rejection of an industry attempt at formulating a dose-response curve demonstrates * * * ," the Court stated, "the Agency's rejection of methods such as dose-response curves was based at least in part on its view tht nothing less than absolute safety would suffice." Id. at 656, n. 63. Such a view is inconsistent with the statutory requirement that OSHA establish a need for more stringent standards.

For this reason, among others, OSHA cannot rely upon the recommendations of the NIOSH CIB to justify workplace ETS regulation. The CIB asserts that ETS meets the criteria for classification as a potential occupational carcinogen under OSHA's Carcinogen Policy. The CIB urges employers to "take steps to reduce exposure [to ETS] to the lowest feasible concentration" and advocates a workplace smoking ban.

The reasoning and recommendation contained in the NIOSH CIB directly contradict the Supreme Court's holding in Benzene. The CIB fails to address the critical issue of whether the putative risk from ETS is significant at workplace exposure levels, even though NIOSH expressly acknowledges the

2024695696

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

lack of quantitative ETS exposure data and the limited information on ETS exposure in the workplace. The CIB simply states that ETS is a carcinogen at some level of exposure and concentrations of ETS should be reduced to the lowest feasible level.[22/]

This is precisely the reasoning struck down by the Supreme Court in Benzene. One of OSHA's premises in setting a PEL of one ppm had been that there was no demonstrably safe level of exposure to benzene. The court rejected the agency's reasoning, finding that OSHA had no evidence that benzene was dangerous at levels of greater than one ppm and less than the prior standard of 10 parts per million. At the very least, Benzene requires that the risk must be "quantified sufficiently to enable the Secretary to characterize it as significant in an understandable way." Benzene, 448 U.S. at 646.

The scarcity of data on workplace ETS exposure, particularly the lack of reliable dose-response estimates, is in sharp contrast to other OSHA standards that have survived judicial scrutiny. In Marshall, for example, the D.C. Circuit upheld OSHA's lead standard because the standard was supported by voluminous data on the harmful effects of lead at various

---

22/ In response to Benzene, OSHA expressly deleted the words "lowest feasible level" from its original Carcinogen Policy. Identification, Classification and Regulation of Potential Occupational Carcinogens; Conforming Deletions, 46 Fed. Reg. 4889, 4890 (1981).

2024695697

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

blood-lead levels and OSHA had correlated those levels with various average air-lead levels.

Similarly, in Asarco, Inc. v. OSHA, 746 F.2d 483 (9th Cir. 1984), the Ninth Circuit sustained the arsenic standard based on a number of large-scale American studies quantifying arsenic exposure levels in the workplace and correlating those levels with worker incidence of cancer. As OSHA emphasized in its Supplemental Statement of Reasons for the Final Rule, "[t]he underlying data upon which the risk assessment for inorganic arsenic are based are high quality epidemiologic studies in an occupational environment." 48 Fed. Reg. 1864, 1895 (1983)(emphasis added). No comparable data exists with respect to workplace exposure to ETS. Because of the lack of ETS workplace exposure data and the insufficient evidence of a dose-response relationship between ETS and lung cancer, there is no legal basis for a standard regulating exposure to ETS.

C. OSHA Cannot Rely Upon Data Extrapolated From Other Sources To Support Regulation of ETS

In the absence of data on workplace exposure, OSHA may not rely upon data either from spousal smoking or active smoking studies to establish a significant risk in the workplace. As in any OSHA rulemaking, OSHA must rely on specific quantitative risk data and not on assumptions and extrapolations taken from other sources.

The Fifth Circuit addressed a similar issue in Texas Independent Ginners Assn. v. Marshall, 630 F.2d 398 (5th Cir.

2024695698

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

1980), when it overturned the cotton gin standard because the evidence did not support a finding of significant risk. The court ruled that OSHA improperly assumed that, because byssinosis results from the high exposure levels to cotton dust found in textile mills, byssinosis also results from the lower exposure levels in cotton gins. The court noted that the cotton ginning industry is characterized by "significantly different conditions and * * * significantly different exposure level[s]" from those that prevail in the cotton textile industry. Id. Assumptions based on studies of one industry do not provide an adequate evidentiary basis for regulation of a different industry. Ibid. In overturning the standard, the court also pointed out that OSHA relied heavily on studies of ginning employees in Egypt, Uganda, Greece and Sudan, rather than on studies of American gin employees, despite clear differences between the exposure conditions in foreign as opposed to U.S. cotton gins. Id. at 408. The Fifth Circuit's holding therefore makes clear that OSHA may not rely on non-U.S. studies as the basis for regulation of the American workplace.

OSHA later amended its occupational standard for cotton dust to eliminate its application to certain segments of the non-textile industry on the ground that the evidence used to support its finding of significant risk in the textile industry did not support such a finding in non-textile operations. Occupational Exposure to Cotton Dust; Final Rule,

2024695699

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

50 Fed. Reg. 51120 (1985). OSHA concluded that its initial assumption that data from the textile industry reflected a significant risk to workers in non-textile operations had overlooked significant differences between the two industries that resulted in lower exposures to cotton dust among non-textile workers, alterations in the composition of the dust to which workers were exposed and, consequently, a lower risk of material health impairment. Without industry-specific studies demonstrating a significant risk from exposure to cotton dust, OSHA concluded, regulations limiting exposure levels in the non-textile industry and mandating implementation of remedial measures were unwarranted.

More recently, OSHA in a Notice of Proposed Rule-making recommended the withdrawal of three substances from the standard governing occupational exposure to asbestos based upon its determination that the evidence supporting a significant risk from exposure to asbestos fibers could not be relied upon as a basis to establish a similar risk for the non-asbestiform substances initially included in the standard. Occupational Exposure to Asbestos, Tremolite, Anthophyllite, and Actinolite, 55 Fed. Reg. 4938, 4943 (February 12, 1990). Those substances, OSHA noted, exhibit substantially different structural and mineralogical traits that could result in different physical responses to their inhalation. Ibid. Although OSHA expressed continuing concern over the health effects of exposure to those non-asbestiform substances, it

2024695700

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

found that conflicting data and deficiencies in exposure information in studies specifically designed to test their carcinogenicity precluded a finding of significant risk. Id. at 4949.

The court's decision in Texas Independent Ginners and these administrative rulings reflect the need for OSHA to rely upon substance and industry specific quantitative risk data rather than data extrapolated from studies involving different substances or industries or non-U.S. studies. Thus, regulation of ETS cannot be based on extrapolation of data from active smoking studies. As explained in Section I, mainstream smoke and ETS are qualitatively and quantitatively distinct substances and exposure conditions differ considerably.

Nor can ETS regulation be based on extrapolation of data from in-home epidemiologic studies to the workplace. ETS exposure conditions in the home differ markedly from workplace exposure conditions. In its denial of Action on Smoking and Health's petition for a standard banning or restricting smoking in the workplace, as well as in the subsequent lawsuit challenging the denial, OSHA repeatedly has emphasized that "the principal reports' heavy reliance on underlying epidemiological studies of spouses' exposure in residential settings limited their applicability" to assessing risk "under actual, prevailing occupational exposure conditions." Brief of the Secretary of Labor, Action on Smoking and Health v.

2024695701

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

OSHA, No. 79-1054 at 13 (D.C. Cir., Mar. 1, 1991).[23/] (OSHA Appellee's Brief). See also Letter from Alan C. McMillan, Acting Assistant Secretary, U.S. Department of Labor, to John Banzhaf, Executive Director, Action on Smoking and Health, at 6 (Sept. 1, 1989).

OSHA further observed that "extrapolation of quantitative risk estimates of occupational risk [of ETS exposure], based even upon reliable estimates of mortality in the general population, is inappropriate. There is simply no agreement as to the likely exposure levels and the degree of risk in workplaces, as distinguished from non-occupational settings. Clearly, categorical assumptions based simply on the percentage of the population in the workforce are not a substitute for reliable data on the extent and variability of exposure." OSHA Appellee's Brief at 24.

Differences between home and workplace conditions are magnified by the fact that many of the published ETS epidemiologic studies relied upon by NIOSH and others were conducted in foreign countries under conditions far removed from the typical American workplace. OSHA therefore may not rely on spousal smoking studies or non-U.S. studies to justify regulation of ETS in the workplace. To do so would require

---

[23/] At oral argument in the same case, counsel for OSHA explained to the D.C. Circuit Court of Appeals that "[t]he principal risk assessments in this are not definitive, because they rely heavily upon data developed in residential studies, rather than on actual occupational exposures." Action on Smoking and Health v. U.S. Dept. of Labor, No. 89-1656, Transcript at 12 (D.C. Cir., May 6, 1991)(oral argument).

2024695702

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

the very kinds of unjustified assumptions and extrapolation of data specifically disapproved by the court in Texas Independent Ginners and rejected by OSHA in its own rulings.

Finally, any regulation of workplace exposure to ETS must be supported not only by data from the workplace rather than the home but by data from each type of occupational setting (e.g., offices versus factories). OSHA already has recognized that conditions of workplace exposure to ETS vary considerably, observing that "actual indoor concentrations may vary widely depending upon employee occupancy, smoker density, ventilation rates and other conditions." Id. at 25, n. 19. As discussed above, however, exposure assessment data is lacking for all types of occupational settings as well as for the workforce as a whole.

D. Mere Irritation or Annoyance Does Not Constitute a "Material Impairment of Health"

In addition to a showing of "significant risk," OSHA must prove that a standard is necessary to reduce or eliminate a "material impairment of health." OSHA may not regulate a substance merely because some individuals find it a source of transient irritation or annoyance. See Texas Independent Ginners, 630 F.2d at 408 (rejecting OSHA's argument that acute effects of exposure to cotton dust such as "daily nonchronic decline in pulmonary function" were sufficient to support cotton gin regulations).

Although the term "material impairment" is not defined by the Act itself, Congress considered and rejected

2024695703

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

language in section 6(b)(5) that would have required standards assuring "that no employee will suffer any impairment of health," S. Rep. No. 1282, 91st Cong., 2nd Sess. 39 (1970)(emphasis added). Congress instead chose to substitute the word "material" for "any." The Act therefore "does not require the Secretary to promulgate standards that would assure an absolutely risk-free workplace." Benzene, 448 U.S. at 647; see also Rothstein, Occupational Safety and Health Law, § 72 at 85 (3d ed. 1990)(suggesting that the term would be given its common workers' compensation definition of "loss of a physical function"). For this reason, OSHA would not be justified in regulating ETS merely because some individuals find it annoying, irritating or offensive.

III. CONCLUSION

Few reliable studies of workplace ETS exposure exist and therefore occupational exposure levels have not been adequately quantified. The available epidemiologic evidence does not support a conclusion that exposure to ETS is associated with an increased risk of lung cancer or other disease. The majority of these studies, moreover, rely on nonsmoker marriage to a smoker as a surrogate for ETS exposure and are of questionable value in assessing potential workplace risk. In any event, none of the U.S. studies reported a statistically significant association between nonsmoker marriage to a smoker and lung cancer, nor have 10 of the 11 workplace studies reported such a risk. It therefore is clear

2024695704

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

that there is an inadequate factual basis for OSHA to regulate workplace exposure to ETS.

In addition, although OSHA has the authority to regulate indoor air quality generally by means of ventilation and similar mitigative measures, the agency itself has emphasized repeatedly that ETS "constitutes one of a number of factors affecting indoor air quality." OSHA Appellee's Brief at 25, n. 19. As studies conducted both by NIOSH and private ventilation experts confirm, the vast majority of indoor air problems are not attributable to ETS. ETS-specific remedial measures therefore are ineffective to alleviate sick building syndrome and other indoor air quality problems. Because of the many variations among workplaces, and because ETS is but one of many factors affecting indoor air, a national standard regulating ETS and not other indoor air substances would not only be unjustified but would fail to remedy the vast majority of indoor air problems.

2024695705

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000

2024695706

Source: https://www.industrydocuments.ucsf.edu/docs/nljj0000